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BOOK REVIEW

The Lung As A Mirror of Systemic Disease, by Eli H. Rubin, M.D.

Charles C. Thomas, Publishers, Springfield, Illinois, U.S.A., 1956

This book is devoted in the main to a description of those diseases in which the respiratory tract is involved in systemic diseases rather than with those which affect the respiratory tract primarily or only. The large task of describing these diseases and the importance of the chest X-ray in their diagnosis is undertaken by the author with, on the whole, good though uneven results.

A thorough review of mass chest X-ray surveys is given in the first chapter with the results analysed and put in perspective. The greatly increased value of such screening techniques in selected situations and population groups, such as general hospitals and private practices is pointed out.

A working classification of general systemic diseases is adopted and they are discussed under separate headings. There are chapters on diagnosis with assessment of the various methods, including history, physical examination, X-ray examination and laboratory and special diagnostic procedures.

The author makes it clear that he is less impressed by physical examination of the chest than most physicians. He feels that the chest X-ray is the keystone of diagnosis of chest disease and that abnormal physical signs, if elicited, are better appreciated after the chest X-ray has been seen and the chest then re-examined. He stresses the limitations of the stethescope as a diagnostic tool. He also stresses however, the limitations of the chest X-ray itself, and states that diagnosis made by X-ray always requires confirmation. With these conclusions, many readers would find themselves at variance.

Some of the chapters on general systemic disease, for example, Boeck's sarcoid, provide a fairly concise and well organized review of the subject including the chest X-ray findings which are of value. Others, for example, the sections on fibrocystic disease of the pancreas, and that on metastatic calcification, are less valuable or inadequate.

The printing is clear and easily read on paper of good quality. The reproductions are of good quality throughout, and this is most obvious where the matter is clinical photography or illustration of pathologic slides. Many of the X-rays reproduced are of unduly high contrast, sometimes also underexposed, and occasionally fogged. The costophrenic sinuses, though often referred to in the text, are cropped in the majority of the illustrations. Organization of the legends is adequate with the occasional exception when the legend is overleaf from its cut.

The bibliography, given at the end of each chapter, is well organized and ample.

In terms of chest diagnosis, this book attempts to be all things to all internists and roentgenologists. In this, it inevitably fails, but it will nevertheless form a useful addition to many libraries and will be helpful in broadening the viewpoint of physicians concerned with chest diagnosis.

J. S. D.

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POLYPOID CHOLESTEROLOSIS OF THE GALLBLADDER: A CLINICAL AND RADIOLOGICAL EVALUATION*

by

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The radiological diagnosis of disease of the gallbladder, perhaps more than any other organ of the body, has been perfected to such a degree that its dependability and accuracy are beyond any reasonable doubt. This accuracy, which must be well over 95 per cent, has developed for two obvious reasons: first, the ease with which the gallbladder may be opacified by relatively non-toxic media and, secondly, its ready accessibility to radiological exploration.

But maybe this knowledge has had the adverse effect of having lulled radiologists and other clinicians equally into a false sense of security. For example, prior to 1955, the diagnoses rendered on the 23 cases of cholesterol polyposis forming the subject matter of this paper would doubtless have been "cholelithiasis in a normally functioning gallbladder." In all likelihood, the result in either case, in so far as the patient was concerned, would have been the same — cholecystectomy if symptoms were severe enough, or conservative therapy if symptoms were mild or easily controlled by diet.

But with most of the 23 cases who were operated upon, the pathologist's report would be likely to list as the main diagnosis, "mild chronic cholecystitis," and then possibly in smaller print beneath, the word "cholesterolosis," with the implication, intended or otherwise, of an incidental finding. Rightly or wrongly, there seems little doubt that in the past this relegation of cholesterolosis to the unimpressive position of an incidental finding, has been due in large part to its being solely a pathological diagnosis. There are few diseases indeed which can claim any degree of clinical prominence which are not possible of pre-operative or premortem diagnosis.

We propose, therefore, to demonstrate that cholesterolosis, in a considerable proportion of cases, is a radiologically diagnosable disease, and that it merits recognition as a potential symptom-producing lesion.

Pathology:

Cholesterolosis is a common pathological entity characterized by an accumulation of abnormal amounts of lipoid, chiefly cholesterol and cholesterol ester, in the gallbladder wall.[1,2,3,4] The deposits are predominantly intra-cellular, partly in the mucosal epithelium where they are arranged in globules in the basal portion of the cells, but more frequently and more abundantly in stromal histiocytes which clump together as foam cells, characteristically aggregated in the tips of the villi. The amount of lipoid deposited varies markedly in degree, producing all gradations from a mild speckling of the mucosal surface with tiny yellow cholesterol granules (hence creating the familiar appearance of an almost ripe strawberry), to a stage where the accumulation of foam cells in the distended villi assumes such proportions as to create large polypoidal masses protruding into the gallbladder lumen (Figure 1).

The former or diffuse variety of cholesterolosis is, and will probably remain, a pathological diagnosis. The morphological changes produced by it are insufficient to cause recognizable alteration in the gallbladder contour as seen radiologically, at least with methods presently at our disposal. It therefore includes the bulk of those cases in which the diagnosis is an incidental finding when the gallbladder is opened after removal.

It is when the local accumulation of lipoid is sufficiently large to distend the villi to polypoidal proportions that cholesterolosis becomes a radiologically significant disease, and it is this group which forms the major subject matter of the present paper.

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*From the Department of Radiology, Royal Victoria Hospital, Montreal, Canada. *Presented at Annual Meeting, Canadian Medical Association, Section on Gastroenterology, June 17-21, 1957, Edmonton, Alberta. Also presented in part as a preliminary report at the Annual Meeting, Canadian Association of Radiologists, Vancouver, January 16-19, 1956, in association with Dr. Albert Jutras and Dr. Henri-Paul Levesque, Hotel-Dieu de Montreal Hospital.*

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That cholesterolosis has been predominantly an operative or pathological diagnosis is obvious from a glance at the rather extensive literature to date.[1,2] In spite of the fact that the accumulation of abnormal amounts of lipoid in the gallbladder wall was recognized by Virchow as far back as 1857,[5] its presence given clinical importance by Moynihan in 1909[6] and the abnormality aptly named "strawberry gallbladder" by MacCarty a year later,[4] it is doubtful if one could find any disease which has been described so thoroughly in the clinical and pathological literature, but which has been so completely neglected by radiologists. In fact, available descriptions of radiological changes are so indefinite and unconvincing as to make their application almost valueless in the interpretation of any one case.[7,8] For an organ which is so accessible to radiological examination it is rather amazing that morphological changes have been almost entirely neglected, while stress has been placed on attempts to assess minor alterations in function, which are so difficult to evaluate and verify. The only exception to this is the now classical work published by Kirklin in 1931 on the radiographic diagnosis of papillomata of the gallbladder.[9] His descriptions and reproductions are almost surely those of cholesterol polyposis, although no mention is made of cholesterol in the article, and the association was apparently not recognized.

Incidence:

During the past two years, members of the Department of Radiology of the Royal Victoria Hospital have observed certain morphological changes in gallbladders which were considered originally to suggest a diagnosis of cholesterolosis and which, on further study, have been found to be almost pathognomonic. Of 23 cases so diagnosed during this two-year period, 17 or almost 75 per cent have been seen in the past six months, a discovery rate which not only points to increased recognition with awareness, but which is also of considerable significance in relation to the overall number of cholecystopathies.

Cholesterolosis, whether occurring alone, or in association with cholecystitis or cholelithiasis is not an uncommon condition. In ten large surgical and autopsy series reported in the literature, its average incidence is 23 per cent of all pathological gallbladders.[1,10,11,12,13] Excluding from this total those gallbladders showing only diffuse cholesterol infiltration — the typical "strawberry" change — as well as those with cholelithiasis or a major degree of cholecystitis, there are left a relatively small but significant number of cases in which a local superabundance of cholesterol has accumulated in the form of polypi.

In two series in which the figures were broken down into pathological sub-groups, polyposis occurred in 26 per cent of all cases of cholesterolosis, or approximately 6 per cent of all pathological gallbladders.[1,11] Surely this figure is significant in relation to the total number of cholecystectomies performed. In addition, there are the not inconsiderable number of cases which, although fairly positively diagnosed on the basis of almost pathognomonic radiologic criteria, are not operated upon because of either insufficient symptoms or the lack of appreciation of the propensity for cholesterol polyposis to rank with cholelithiasis or chronic cholecystitis as a disease whose symptoms may benefit by cholecystectomy.

Pathogenesis:

To delve into all the various theories of the pathogenesis of cholesterolosis in any detail would be not only impractical but superfluous, especially since controversy still exists over many of its aspects. Briefly, pathogenesis resolves itself into three main considerations:

1. The source of the cholesterol;
2. The cause of its accumulation in increased amounts in the gallbladder wall; and
3. The relationship between cholesterolosis and cholelithiasis.

To be able to answer all of these would be to bring one close to a solution of the pathogenesis of all gallbladder disease. Unfortunately, the answers are only theoretical at present, and include the following most widely accepted concepts:

The normal gallbladder mucosa is capable of absorbing not only water but also a variety of lipoids and even particulate matter.[1,2,3,14,15,16] The amount of lipoid absorbed, particularly cholesterol and its esters, depends largely on its concentration in the bile. The gallbladder bile of patients with cholesterolosis is characteristically thick, tarry and supersaturated, the proportion of cholesterol having been estimated by Illingworth at twice that of normal controls.[1] The only other source of cholesterol is the blood, and repeated examinations have shown no greater incidence of hypercholesterolaemia in those patients with cholesterolosis than with other forms of gallbladder disease.[1] There seems little doubt, therefore, that the cholesterol comes from the bile. So much for the source.

Is the increased amount of cholesterol passing through the gallbladder wall sufficient to explain its deposition? It seems unlikely, for a curious change takes place in the character of the lipoid

in cholesterolosis which is not seen in the normal gallbladder. Under normal circumstances, neither cholesterol nor its esters are identifiable in the gallbladder wall by histochemical or staining methods. In cholesterolosis, some change occurs in the physical and chemical properties of the lipoid so that it becomes identifiable as cholesterol and its esters, by reason of their property of rotating polarized light.[1,3] We thus have a rather shaky foundation on which to support a theory of abnormal deposition.

But why should this physical and chemical alteration occur ? On the answer to this question hangs the "raison d'être" of cholesterolosis. The most logical explanation to date was originally suggested by Chiray and Pavel,[17] who theorized that inflammation, even of mild degree, could alter not only the character of the lipoid itself but also the processes which normally rid the gallbladder wall of these substances. There is much to be said in favour of this theory statistically.[1,2] The majority of cases of cholesterolosis show varying degrees of co-existing cholecystitis, much more frequently mild than severe. In fact, it is important to note that the degree of inflammation generally bears an inverse ratio to the amount of cholesterol — the more the cholecystitis, the less the cholesterolosis.[2,18,19,20]

Finally, what relationship exists between cholesterolosis and gall stones ? Statistically the evidence is overwhelming in favour of a close association, most figures quoted in the literature indicating co-existence in well over 50 per cent.[2,11] On the other hand, in a recent study of over 1300 consecutive adult autopsies, Feldman and Feldman[10] found cholelithiasis in only 18 of 165 cases of cholesterolosis, an incidence of 10.9 per cent. Their conclusion that the incidence of gall stones in cholesterolosis is lower than in the general autopsy population is at variance with other reported series, the difference undoubtedly arising from the expected variation between autopsy and surgical series. Almost without exception when stones are present they are of the cholesterol-rich type, solitary cholesterol and mulberry stones occurring with practically equal frequency. Two theories have been suggested for the formation of calculi:

1. Fat-laden polypoid masses arising from the mucosa are commonly attached by thin, filamentous stalks which may become detached to form a nidus about which cholesterol could be deposited. This theory, originally proposed by Lichtwitz[21] and supported by Boyd[3] and other observers, takes little heed of the fact that remnants of soft tissues are rarely identifiable in the centre of cholesterol calculi. One practical aspect of this ready detachability, however, lies in the ease with which such polypi may be dislodged during rough handling of the gallbladder in the operating room, to the discomfiture of the Radiologist when shown a specimen whose mucosa seems as smooth and normal as a piece of velvet !

2. A more plausible explanation for the frequency of co-existence of cholesterolosis and calculi lies with the very pathogenesis of cholesterolosis itself — a supersaturated bile.[1,2] It is not difficult to conceive the ease with which cholesterol might be deposited out of a saturated solution onto small masses of particulate material, but if this theory is indeed tenable, it is perhaps surprising that the co-existence of the two is as low as 50 per cent.

In essence, however, it seems likely that no direct cause-and-effect relationship exists between cholesterolosis and calculi, but that the two are intimately linked in a common pathogenesis.

Clinical Manifestations:

The symptomatology of cholesterolosis, whether of the diffuse or polypoid variety, differs in no fundamental respects from that of chronic cholecystitis or cholelithiasis. The fact that differentiation on clinical grounds is never really possible is clearly in evidence in several reviews of the subject that have appeared in the literature.[1,2,10,18,22,23,24] Perhaps the most comprehensive of these is a study of 1000 selected cases of cholesterolosis by Judd and Mentzer of the Mayo Clinic in 1927,[12] all diagnoses being confirmed pathologically following cholecystectomy (Table I). Five hundred of the gallbladders showed varying degrees of cholecystitis as well as cholesterolosis but were stone-free; the other 500 had

1000 CASES OF CHOLESTEROLOSIS

JUDD & MENTZER, MAYO CLINIC, 1927.

(26% of all Cholecystectomies over 4 years)

500 GALL BLADDERS - WITHOUT CALCULI (CHOLECYSTITIS ±)

500 GALL BLADDERS - WITH CALCULI — SINGLE 30% / MULTIPLE 70% — 99% CHOLESTEROL-RICH

DIFFUSE ("STRAWBERRY")	- 53%	INCIDENCE
POLYPOID	- 47%	SEX — 80% FEMALE
COEXISTING (STONE-FREE)	- 34%	AGE — 35 - 40 MAX.

	SYMPTOMS	
	STONE-FREE	CALCULI
ACUTE BILIARY COLIC	35%	60%
SEVERITY OF PAIN	++	+++
"DYSPEPSIA"		
R.U.Q. PAIN	65%	40%
INDIGESTION	All Patients	All Patients
JAUNDICE	17%	17%

TABLE I

cholelithiasis in addition. The incidence of cholesterolosis was found to be 26 per cent of all gallbladders removed, and of these the diffuse strawberry change was present in 53 per cent and the polypoid variety in 47 per cent. The two co-existed in 34 per cent of the stone-free cases. This high incidence of polyposis is indeed striking, and is greater than that reported in most series. Clinically, there was observed a female sex predominance of 3:1, the difference being ascribed chiefly to pregnancy and obesity.

Symptomatically, little difference was observed between the cases with or without calculi. *Acute attacks* of severe biliary pain were less frequent in the stone-free cases, being recorded in 35 per cent as compared to 70 per cent of those with calculi. *Pain* was

felt to be generally less severe, so that relatively fewer patients without stones required morphia for relief. *Indigestion* was a universal complaint in both groups, symptoms of vague epigastric and right upper quadrant pain, fat intolerance, flatulence and bloating being of about equal incidence in the two groups. Surprisingly, *jaundice* was observed as frequently in those cases without stones as in those with, amounting to 17 per cent of each group. This high incidence of jaundice in patients whose gallbladders are stone-free has had no completely satisfactory explanation, although cholangitis, stenosis or kinking of the common duct, spasm and oedema of the ampulla of Vater and a passed stone are all suggestions which seem to bear some merit.

The clinical findings in the 23 patients who form the basis of this report (Table II) differ in no fundamental respect from the large series reported by Judd and Mentzer. Although all are examples of the polypoid variety of cholesterolosis, this factor seems to be important only from the standpoint of preoperative diagnosis, since the symptomatology of the polypoidal group appears to differ from the "strawberry" group only in degree, as might be expected. In fact, Judd and Mentzer make no mention of a significant variation in the severity of the symptoms of the two.

23 CASES OF CHOLESTEROLOSIS

R. V. H. - 1955-1957

ALL POLYPOID (WITH OR WITHOUT "STRAWBERRY" CHANGE)

NONE WITH CALCULI

ACUTE BILIARY COLIC	7 (30%)	ALL BUT ONE HAD CHRONIC DYSPEPSIA
DYSPEPSIA	17 (74%)	
ASYMPTOMATIC (OR NON-BILIARY)	5 (22%)	
JAUNDICE	0	

TABLE II

Six, or 27 per cent of our 23 patients had attacks of typical severe right upper quadrant pain of biliary origin. In addition, 16 (70 per cent) had symptoms of chronic "gallbladder dyspepsia" of varying duration, generally over a period of several years. Seven patients were either asymptomatic or had complaints which were so indefinite and atypical that they could not be seriously ascribed to gallbladder disease.

One might well ask what is the cause of the symptoms in cholesterolosis? From the many discussions which have appeared in the literature since 1909, when Moynihan first described it as a disease requiring cholecystectomy,[6] this question has been the subject of much dispute. Several observers, of whom Mackey is perhaps foremost, have insisted that cholesterolosis is merely one local manifestation of a general metabolic disturbance and as such is unlikely to produce symptoms.[2] Others, Illingworth for example, believe that symptoms are invariably the result of the associated cholecystitis, and that when inflammation does not exist, neither do symptoms.[1] The frequency of co-existing cholecystitis lends some support to this thesis, in spite of the almost inevitable mild degree of inflammatory change which by itself would hardly be expected to cause symptoms. The conclusion that cholelithiasis must co-exist before symptoms are produced, as propounded by Womack and Haffner,[18] is untenable, not only as revealed by the present series, but also by the large group reported by Judd and Mentzer. Womack's attractive theory as to the cause of symptoms in all forms of gallbladder disease seems to have some application in cholesterolosis.[23] He found inflammatory changes around sensory-nerve endings in the wall of the gallbladder, and proposed that pain sensations might arise from these overexcitable nerve-endings from stimuli such as stretching, which would ordinarily cause no response in a non-inflamed wall. He further suggested that if such a change influences sympathetic nerve fibres in this fashion, it is reasonable to suppose that parasympathetic motor fibres might respond in the same way so as to produce increased irritability of the gallbladder, a manifestation frequently seen in cholesterolosis.

The importance of so-called biliary dyskinesia as the fundamental cause of symptoms, not only in cholesterolosis but in other form of gallbladder disease as well, undoubtedly deserves further study and amplification. The work of Dedeu and Braier in investigating common duct pressure manometrically in cases of gallbladder disease, points to spasm or oedema of the sphincter of Oddi or the cystic duct as a potentially frequent culprit in the pathogenesis of gallbladder symptoms, these alterations presumably being secondary to the cholecystitis itself.[24]

Radiological Manifestations:

The radiological manifestations of cholesterol polyposis are as might be imagined from the gross appearance of the mucosal

surface of the opened gallbladder. What would otherwise be regarded as a perfectly normal mucosa is studded with several pedunculated masses varying from 3 to 10 mm. in diameter. Although occasionally solitary, they are almost always multiple, as many as 10 or 12 being scattered irregularly over the mucosal surface. Characteristically, they are more abundant in the upper portions of the body and infundibulum than in the fundus, and tend to be irregular in shape but always covered by an intact mucosa. Their pedicles are frequently thin and tenuous, so that even minor trauma may detach them from their moorings (Figure 1). An associated diffuse involvement of the remaining mucosa frequently produces a "strawberry" appearance, although a considerable proportion of cases will show polypi only.[1,11]

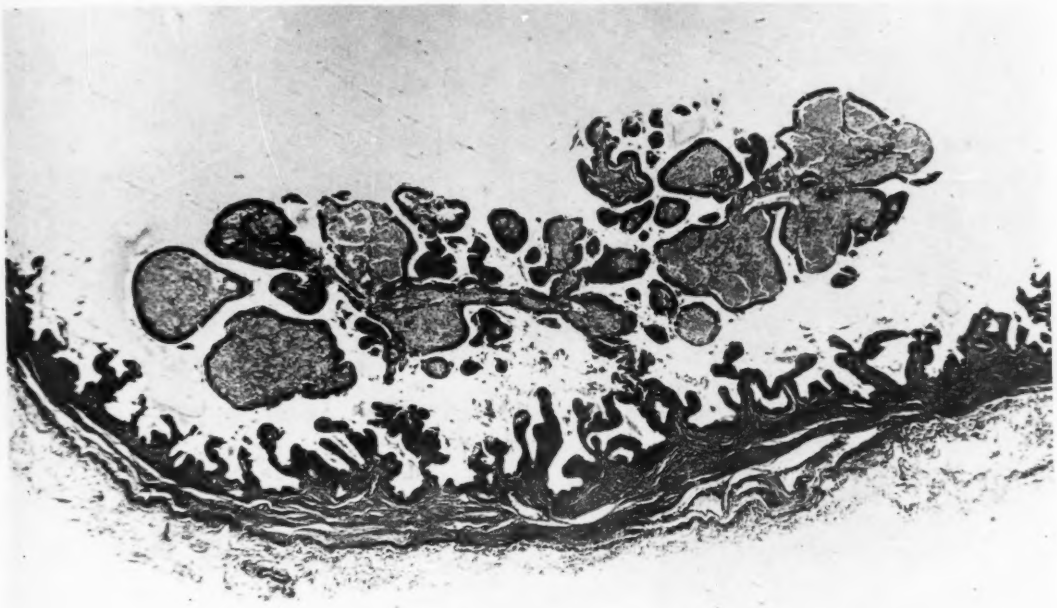

Fig. 1 — Low power photomicrograph of the gallbladder wall of a representative case, showing a large cholesterol polyp arising from normal mucosa. The polyp possesses an intact mucosal covering and is filled with lipoid-laden macrophages. *Note* the extremely thin tenuous stalk.

It is easy to understand how the normally smooth, sharply delineated contour of the healthy gallbladder, as seen cholecystographically, could be deformed by such lesions. Protruding into the dye-filled lumen are defects of varying size, almost always discrete but occasionally confluent (Figure 2). Their contour is irregular and commonly less sharply defined than calculi. In contrast to calculi they remain constant in site, whether the patient be examined in the erect or supine position. These are perhaps the most important single factors in the differentiation from small radiolucent calculi. Visualization in profile is important, especially in the study of small polyps since, in further contrast to calculi, no dye-containing bile will separate the lesion from the wall (Figure 3).

As in the identification of small radiolucent calculi in a gallbladder whose functional capacity is maintained and concentration of dye is therefore dense, visibility of small polypi is always enhanced following the partial emptying of the gallbladder in response to a fat meal (Figure 4 and 5). Oftentimes, several polypi will be identified on "p.c." films which were not readily visualized in the filled state, especially since cholesterolosis is seldom associated with any reduction in the concentrating ability of the gallbladder.

It is assumed that our procedure for cholecystographic examination is similar to that employed elsewhere. It need hardly be stressed that compression studies in the erect and supine positions under fluoroscopic control is a *sine qua non* of adequate diagnosis. Time and again defects have been visualized on pressure spot-films which were not visible on routine overhead films. All patients in this

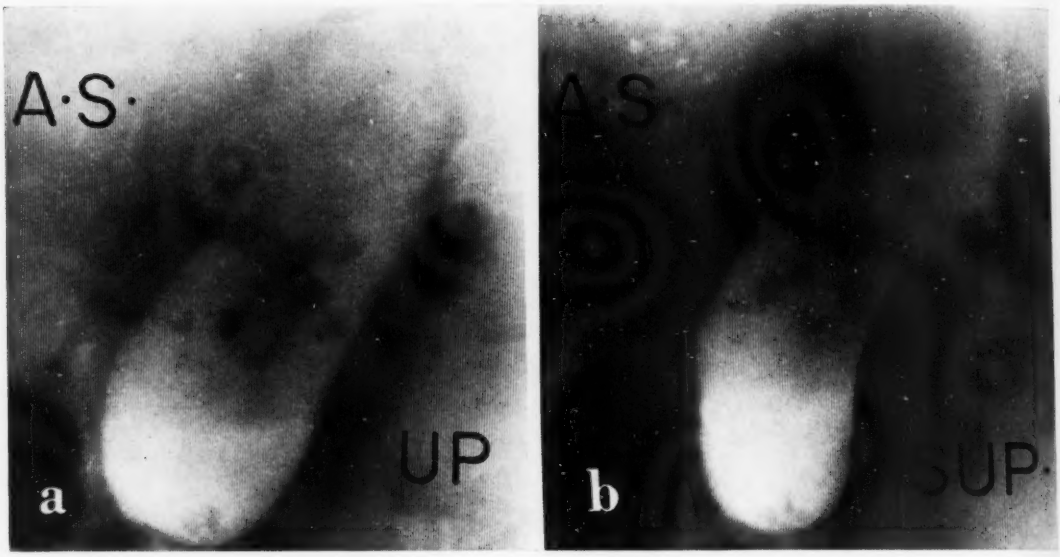

Fig. 2 — Roentgenograms of the gallbladder before a fat meal in the (a) erect and (b) supine positions. *Note* the multiple defects in the dye-filled lumen, possessing the following characteristics:

(1) irregular in contour and rather indistinctly defined;

(2) constant in site in both positions;

(3) more abundant in the upper portion of the body and infundibulum, there being only one in the fundus;

(4) the absence of any tendency for the defects to "layer" with the erect posture. *Note* also that the density of the concentrated dye is at least average, if not greater than average.

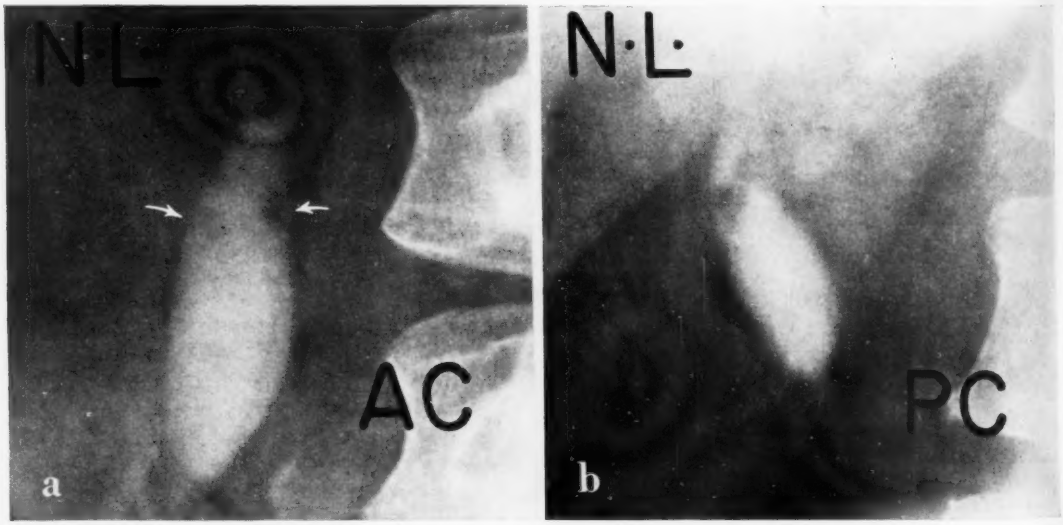

Fig. 3 — Roentgenograms in the erect position, (a) before and (b) after a fat meal. *Note* —

(1) better visibility of the two polypoid defects (arrows) after partial evacuation than before;

(2) the continuity of the wall of the gallbladder and the lesions, indicated by lack of interposed opaque media;

(3) The absence of any change in position of the lesions in relationship to each other, or relative to the fundus and cystic duct.

series have been examined by the oral method (using Telepaque(R) as the cholecystographic media). Our experience with intravenous cholangiography in this particular disease is not yet sufficient to allow any definite conclusions to be drawn.

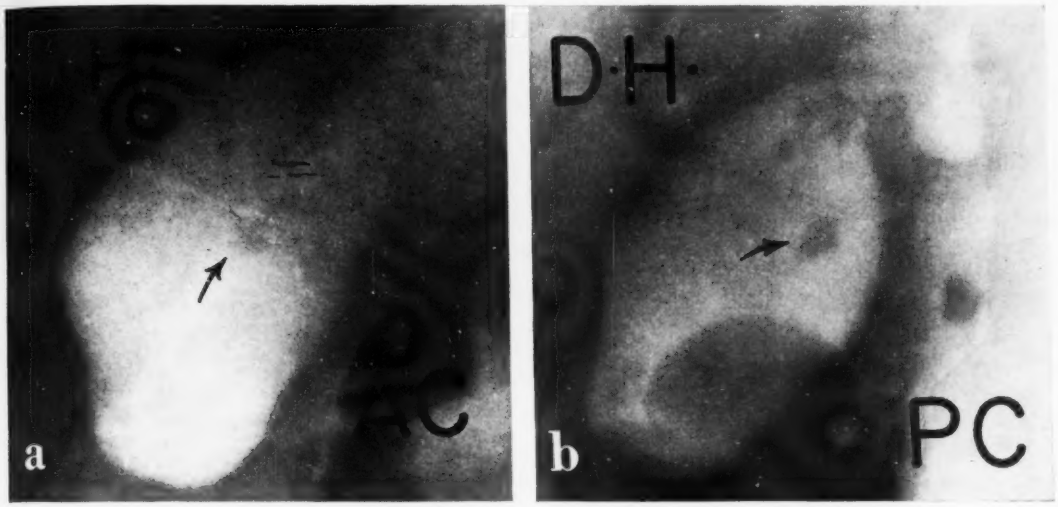

Fig. 4 — Roentgenograms in the erect position (a) before and (b) after a fat meal. *Note* —
(1) only one defect is seen on *both* radiographs (arrow);
(2) after partial evacuation of bile in response to fat, several more defects become visible in the infundibulum (b).

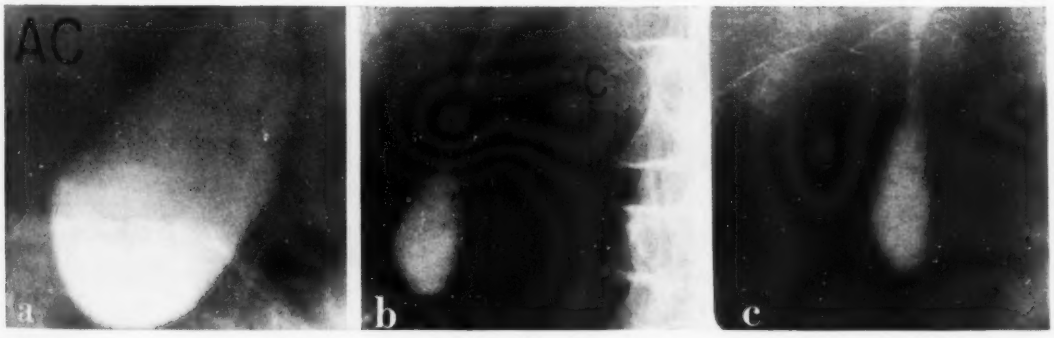

Fig. 5 — Roentgenograms (a) before and (b and c) 10 minutes after a fat meal, all exposures being made in the erect position with the pressure "spot film" device. *Note* —
(1) the better visibility of the multiple small defects after partial evacuation of bile than before;
(2) their tendency to be grouped in the upper body and infundibulum;
(3) the remarkably rapid response to a cholegogue;
(4) average or greater than average density of concentrated dye-laden bile.

The morphological changes just described form the major part of the radiological story, but not the whole of it. It stands to reason that in the final analysis we must depend on morphological alterations as being the only changes on which a definitive diagnosis of cholesterolosis can be based. Cholecystography, however, is partly also an examination of the functional capacity of the gallbladder, and it is upon this aspect that brief comment should be made.

One might assume from the minor alteration of the gallbladder wall which takes place in cholesterolosis that little change in the function of the organ should occur, and this is indeed true. It has been our observation, as well as that of others,[8] that the concen-

trating power of the mucosa is not only maintained but is in many cases greater than average, with the result that the density of the gallbladder shadow is striking (Figures 2 and 5). A similar and equally frequent observation, albeit one subject to the inherent error of personal evaluation, is the apparent increased rapidity and degree to which the gallbladder empties in response to fat (Figure 5). Although considerable conflict of opinion exists in the literature in this respect, there seems little doubt that the majority of gallbladders with cholesterolosis exhibit moderate irritability, not only emptying more rapidly than normal, but more completely. It is not infrequent to see a marked reduction in size within 15 to 20 minutes after a fat meal.

Differential Diagnosis:

The *differential diagnosis* of cholesterolosis includes only three conditions of any significance, and in only one of these need any great difficulty of differentiation be anticipated. *Radiolucent calculi* are almost never constant in position, varying with change of posture and frequently "layering" in the upright position. Although occasionally marginal, they will always show a thin layer of dye separating them from the gallbladder wall, a separation rarely seen with polypi (Figures 6 and 7).

Fig. 6 — Roentgenogram exposed in the supine position after a fat meal. There are not only several large defects arising from opposing surfaces of the fundus and from the lateral wall of the infundibulum, but also a large central radiolucent shadow in the body. None of these showed change in position from the erect to the supine positions, but the presence of dye-laden bile separating the large central lesion from the gallbladder wall establishes it as a calculus, whereas the marginal position of the others indicate their polypoid character.

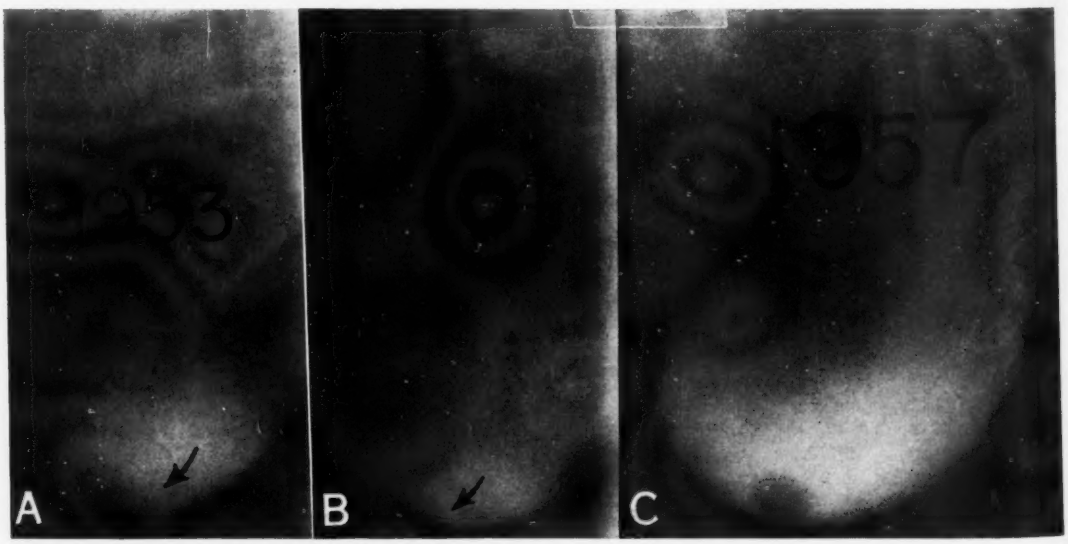

Fig. 7 — Roentgenograms made over a four-year span, (a) and (b) being in the erect position and (c) in the supine (no change was noted in the position of the fundal defect in 1957 with films made in the erect posture). The growth of the lesion (arrows) over a four-year period gave rise to some concern about the possibility of a true papilloma. Cholecystectomy was performed, and a solitary calculus measuring 1 cm. in diameter was found in the gallbladder; there were no polypi. Greater attention should have been paid to the thin line of dye separating the defect from the gallbladder wall.

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A much rarer abnormality, but one in which differential diagnosis from cholesterol polypi may be difficult and at times impossible, is the *papilloma* or *adenomatous polyp* of the gallbladder. The rarity of these true benign neoplasms (as indicated by the incidence of 1 in every 100 cholecystectomies in Shepard's series[25]), has only recently been realized. This is undoubtedly due to an unfortunate confusion of terminology recurring in the literature for many years, the word *papilloma* being used interchangeably with *polyp*. To quote from a recent paper by

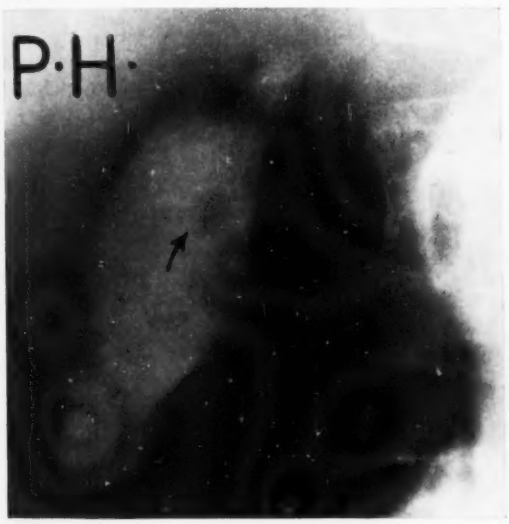

Fig. 8 — Roentgenogram showing a solitary defect in the mid-portion of the body, seen *en face*, and constant in site no matter in what position the patient was examined. Although its site was atypical, the fact that it was solitary suggested the possibility of a *papilloma*. This diagnosis was finally established pathologically.

Tabah and McNeer:[26] "the term *papilloma* should be restricted to those lesions which are truly neoplastic and which represent in the gallbladder the same type of tumour which we find more frequently in the urinary bladder and the gastrointestinal tract."

It is perhaps because of failure to adhere to this well-established pathological principle that cholesterol polyposis of the gallbladder has received the meagre attention it has, particularly with reference to Kirklin's classical description of the radiographic features of what he termed "papillomas" of the gallbladder.[9,27] By virtue of the morphological similarities of cholesterol polyps and true benign papillomata, it is easy to appreciate how difficult differentiation of the two might be radiologically, especially since papillomata are more frequently multiple than single.[25] Only when they are single, under which circumstances they are frequently fundal in position, may the diagnostic stress be placed on *papilloma* (Figure 8). The importance of differentiation is obvious, however, when it is realized that recent reports indicate a strong tendency for benign papillomata to undergo malignant degeneration.[25,26,28]

Brief mention only need be made to one other, even more rare condition which, again, has given rise to some confusion of terminology. On occasion, inflammation of the gallbladder wall gives rise to a local active proliferation and hyperplasia of the mucosal epithelium so as to produce a papillomatous out-pouching from the mucosa. The descriptive term "cholecystitis glandularis proliferans" was applied to this lesion by King and MacCallum.[29] By dint of being almost always solitary and possessed of a broad

Fig. 9 — Roentgenogram showing a solitary, poorly defined but constant defect in the fundus, possessing the usual characteristics of a polypoid lesion. Pathologically, the wall of the gallbladder was locally thickened but without gross evidence of a polypoid lesion arising from the mucosa. Histologically the diagnosis was *cholecystitis glandularis proliferans*, localized to the fundus. The remainder of the gallbladder showed only mild chronic cholecystitis. The broad base of the lesion seen cholecystographically might have suggested the possibility pre-operatively, but is not in itself a diagnostic feature.

sessile base (Figure 9), it should offer little difficulty in differentiation from cholesterol polyposis, but seldom can be differentiated pre-operatively from true neoplastic papilloma.

The diagnosis of cholesterolosis is therefore primarily a radiological problem, assuming that the morphological changes are chiefly polypoidal in nature. One other diagnostic procedure occasionally mentioned in the literature as being of some value, is duodenal drainage. Since cholesterol has been found to be constantly elevated in the "B" bile in cholesterolosis, its diagnostic potentialities are obvious, but it is felt that its use might be restricted to those cases in which the presence of cholesterolosis is suspected but radiological changes are absent.

Treatment:

Whatever the cause of the symptoms in cholesterolosis, their cure by removal of the offending organ remains as the best judge of their origin, and on this aspect available evidence is more convincing (Table III). Of the 23 patients diagnosed as cholesterol polyposis during the past two years at the Royal Victoria Hospital, 10 were operated upon and cholecystectomy performed in 9 (the gallbladder of the 10th case *felt* normal to the surgeon on palpation and was therefore not removed). Of these 9, the 6 who had experienced acute attacks of severe biliary pain have all been completely well up to 2 years after operation. Of the remaining 3, two had had no acute attacks, complaining only of mild chronic dyspepsia, and in neither of these has there been any improvement. The ninth was asymptomatic but was operated upon because of the question of possible malignant degeneration of true papillomata.

TREATMENT

JUDD & MENTZER			ROYAL VICTORIA HOSPITAL	
FOLLOW-UP - 5 YEARS			FOLLOW-UP UP TO 2 YEARS	
(NOT ALL 1000 CASES)			10 PATIENTS OPERATED UPON:	
			8 CHOLECYSTECTOMIES 1 AUTOPSY 1 NO CHOLECYSTECTOMY	
RESULTS			RESULTS	
	GOOD	POOR		
STONE-FREE	87%	8%	5 ACUTE COLIC + CHRONIC DYSPEPSIA	4 WELL 1 IMPROVED
WITH CALCULI	96%	2%	1 CHRONIC DYSPEPSIA	DEAD (CANCER)
(NO BREAKDOWN AS TO RESULTS WITH COLIC AND NON-COLIC PATIENTS)			2 ATYPICAL SYMPTOMS (Chronic Neurosis)	UNIMPROVED
			1 ASYMPTOMATIC	UNCHANGED

TABLE III

These figures are duplicated percentage-wise in almost all reports appearing in the literature.[1,2,6,13,22,23,24,30,31] Keeping in mind the variation in criteria for cholecystectomy used by different observers, the results in all series are in direct proportion to the percentage of patients presenting with a history of severe biliary pain. Whether the symptom-producing disturbance in cholesterolosis is a functional derangement or organic, cholecystectomy will almost inevitably produce a cure in those patients who experience "colic". If, on the other hand, symptoms are vague and ill-defined, the results of cholecystectomy are almost universally bad. It seems possible that, in these cases, symptoms are arising from outside the biliary tract altogether.

Summary and Conclusions

1. Cholesterolosis of the gallbladder is not an uncommon abnormality. Its average incidence in reported series is 23 per cent of all pathological gallbladders. Although its pathogenesis is not fully understood, the most currently accepted theories have been briefly outlined, and consist of two fairly constant alterations:

(a) a highly concentrated supersaturated bile; and

(b) a frequently associated although usually mild cholecystitis.

The relationship of cholesterolosis to cholelithiasis seems fairly firmly established, probably on the basis of similar pathogenesis and not cause-and-effect.

2. Although the literature contains a plethora of reports on the pathology of cholesterolosis, discussions on its radiological characteristics have been almost entirely restricted to functional changes without regard to morphological alterations.

3. Within the past few years at the Royal Victoria Hospital, certain morphological changes have been observed in gallbladders examined cholecystographically which have been confirmed repeatedly as due to cholesterolosis. The multiple irregular polypoid elevations of the mucosa seen pathologically and possessing a diameter from 3 to 10 mm., produce scattered filling defects in the dye-filled lumen of the gallbladder. These changes represent polypoid cholesterolosis and are the most frequent radiological manifestation of the disease.

4. In addition to these morphological changes, on which the majority of diagnoses are based, there are certain secondary signs which are primarily functional. Previously described in the literature as being the only radiological findings in the disease, they have been confirmed in our cases, and are, namely:

(a) Maintenance of excellent functioning capacity of the mucosa, manifested by dye concentration which is as great as and often greater than normal.

(b) An increased irritability of the gallbladder, manifested chiefly by a more rapid response to fat than is normally seen.

5. The differential diagnosis should offer no great difficulty if the typical characteristics described are taken into consideration. It should be stressed, however, that since papillomata of the gallbladder, representing true neoplasms, are more frequently multiple than single and show a propensity to undergo malignant degeneration, differentiation of cholesterol polyposis from this condition is most important, although often impossible.

6. Clinically, the symptoms and signs of cholesterolosis, even when uncomplicated, are indistinguishable from chronic cholecystitis and cholelithiasis. They consist primarily of recurrent acute attacks of severe biliary pain, and chronic "gallbladder dyspepsia."

7. Cholecystectomy is the treatment of choice in two groups of patients: those who present with a history of biliary colic; and those in whom differentiation from multiple papillomata and therefore possible malignancy is impossible. Patients whose symptoms tend to be vague and ill-defined do not as a rule benefit from cholecystectomy.

8. Finally, in conclusion, it is felt that uncomplicated polypoid cholesterolosis is, in the majority of instances, a radiologically diagnosable disease, and that its diagnosis is of major importance because of its potential as a symptom-producing lesion.

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LOCALIZED OSTEOCHONDRITIS OF THE SPINE*

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The purpose of this paper is to draw attention to a condition which is not rare but which is important, and frequently misdiagnosed by radiologists. Osteochondritis of the dorsal spine (Scheuermann's Disease) presents a characteristic and widely recognized clinical and radiological appearance. In this condition the X-ray changes seen are so typical that they are not likely to be mistaken for any other condition.

There is however, a localized form of this disease usually involving only one or two vertebrae, which is less well known. It occurs in the dorso-lumbar region. Its radiological appearance very frequently leads to an erroneous diagnosis of tuberculosis of the spine. Following some introductory remarks regarding Scheuermann's Disease, three recently observed cases of the localized form of this interesting disease will be presented.

Scheuermann's Disease

Osteochondritis affecting the dorsal spine (Scheuermann's Disease) was first recognized by Scheuermann in 1921, and was found to produce symptoms in young persons between the ages of twelve and seventeen. Both sexes are equally affected. In this condition there is kyphosis associated with fragmentation of the epiphyseal ring, progressive narrowing of the intervertebral disc space, deep irregularities on the edges of the vertebral bodies, Schmorl's nodes and anterior wedging of the vertebrae. These changes are usually located in the lower dorsal and upper lumbar area (Fig. 1). Scheuermann called the condition kyphosis deformans juvenilis, believing it to be osteochondritis and therefore similar in etiology to Perthe's disease in the femur and Kohler's disease of the tarsal scaphoid bone.

Symptoms: Pain is usually present and most patients complain of a "tired feeling" in the back. The pain is characteristically aggravated by long periods of standing, and is relieved on lying down.

Ossification: Brief consideration of the ossification of the vertebrae is essential to an understanding of the disease. A typical vertebra ossifies from primary and secondary centres. Primary centres are two in number—one for each side of the neural arch (appearing at the seventh or eighth week) and one for the body (appearing at the eighth week). The arch centres fuse between one and seven years. Fusion between arch and body occurs in the cervical region in the third year, in the lumbar region in the sixth year.

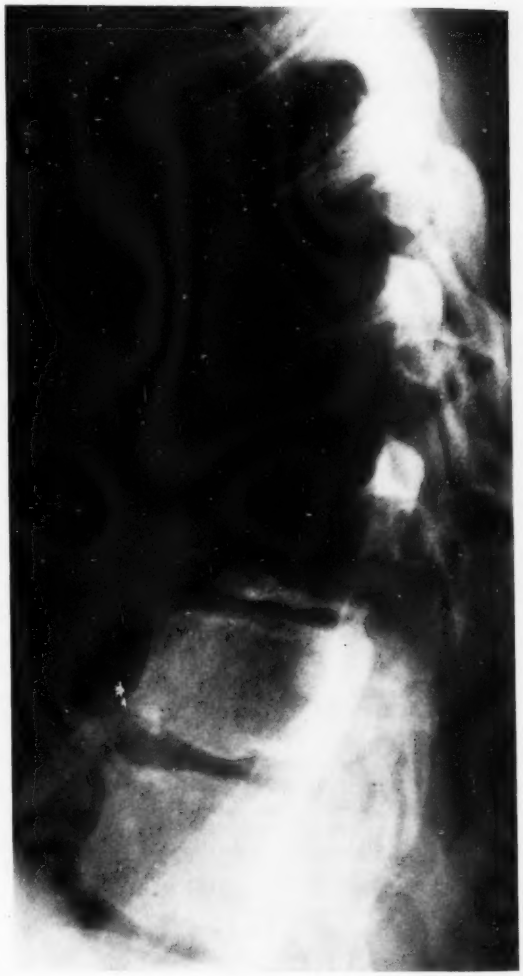

Fig. 1: Scheuermann's disease.

Secondary centres appear at or about the age of puberty. They are as follows — one for each transverse process; one for each spinous process; one for each articular process

*Presented at a meeting of the McGill Reporting Society held on November 5, 1956, Montreal.

(superior and inferior); one each for the superior and inferior rims of the body. Normally all these epiphyses are firmly united at twenty five years of age.

Ossification of the first, second and seventh cervical vertebrae follows a slightly different pattern which need not be discussed here.

Etiology: Scheuermann believed that the "epiphyseal rings" contribute to longitudinal growth of the vertebral bodies. Trauma to these cartilaginous marginal rings, in Scheuermann's opinion, causes ischemic necrosis of the rings and therefore a disturbance in epiphyseal growth.

According to Schmorl, however, longitudinal growth of a vertebra is exclusively the function of the cartilaginous plates, which are the counterparts of the proliferating cartilage and provisional zones of calcification in tubular bones. It is Schmorl's belief that the "epiphyseal rings" lie outside the zones of growth in the vertebral bodies, external to the growing cartilaginous plates. Ehrenhaft[3] and Bick and Copel[1] agree with Schmorl's concept in this regard. These authors all believe that excessively heavy stresses on the articular plates cause the nucleus pulposus to break through the plates into the vertebral bodies. This may occur at several sites in different bodies or in a single body. It produces uneven growth and marginal defects. The load on the vertebral body shifts toward the ventral segment where growth becomes retarded. Wedging develops, followed by kyphosis. Fragmentation of the "epiphyseal ring" is a secondary compression phenomenon, according to this hypothesis.

It is interesting to speculate regarding the nature of the traumatic stresses mentioned by these authors. Some stresses are very likely due to vigorous athletics. Heavy manual labour is mentioned as a cause of trauma but is not done by most children in this age group. The lesions often occur in children whose activities have been normal, and not strenuous in any respect. In these instances Schmorl has suggested that the cartilaginous plates may be congenitally weak.

In the opinion of Caffey[2], there is little evidence to sustain Scheuermann's hypothesis. He believes that many cases must be due to the mechanism described by Schmorl, but admits that there is frequently no radiological evidence of injury to the cartilaginous ring or to the intervertebral disc.

An interesting theory concerning the etiology of spinal osteochondritis is that of Kemp and Wilson[5]. They believe that malnutrition is an important contributing factor. This may be on the basis of poverty, long-standing illness or feeding difficulties. These authors consider that malnutrition during growth years may cause osteochondritis, also malformation of the teeth and dental caries in these children.

Probably Scheuermann's syndrome can develop from more than one pathogenetic mechanism.

Localized Osteochondritis of the Lumbar Spine

This is a condition which seems to be of the same nature as Scheuermann's disease in the dorso-lumbar spine. A report by Hafner[4] in 1952 described four cases. In 1954 Lamb[7] reported seven additional carefully documented cases, all of which were considered to be localized vertebral osteochondritis. Lamb's report is the most complete and comprehensive contribution to the literature of the past ten years, and will be considered in some detail.

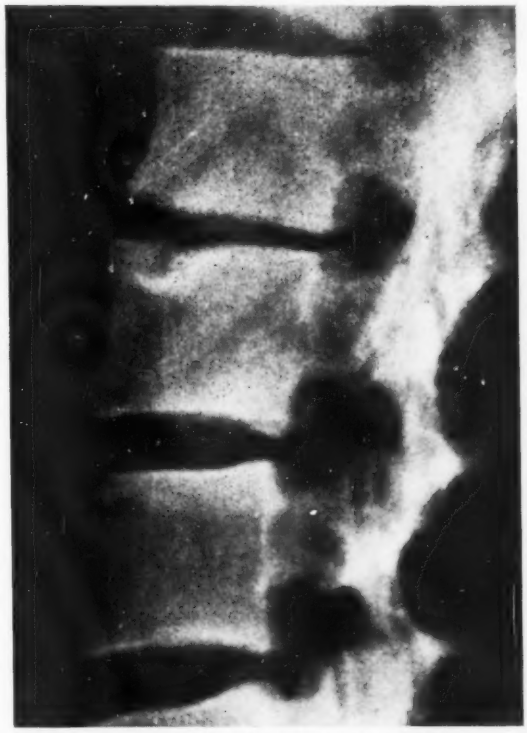

Fig. 2*: Localized osteochondritis.

The seven cases he described all showed defects involving the upper or lower anterior corner of a lumbar vertebra or vertebrae.

**Originally reproduced in Journal of Bone and Joint Surgery, 1954, 36B.*

There was frequently narrowing of a disc space. In some instances, Schmorl's nodes were seen. In four cases only one vertebra was involved; two patients showed involvement of two vertebrae and in one instance a single lumbar body was diseased and slight associated dorsal changes were noted. One of Lamb's cases, showing involvement of a single lumbar vertebra, is illustrated in Fig. 2.

All seven patients complained of pain, six in the lumbar spine and the other in the region of the hip. Three showed spasm of paravertebral muscles with restriction of movement. Lamb's hypothesis concerning the characteristic anterior defect is as follows. There is weakness of the disc, either congenital or post-traumatic. As the fluid nuclear material escapes, the disc space narrows. The fluid material may break the cartilage plate to form Schmorl's nodes, but *may* exert pressure anteriorly. The anterior longitudinal ligament resists this force, which is directed onto the upper or lower corner of the vertebral body. There is consequent pressure necrosis of bone and formation of the characteristic defect.

Differential diagnosis from tuberculosis is important. All the lesions in Lamb's series of cases had been diagnosed by competent radiologists as probably being tuberculous. The following are significant factors in the differential diagnosis. (1) There is no paravertebral abscess formation. (2) The vertebral defect has a characteristic "punched out" appearance. (3) Schmorl's nodes or other evidence of vertebral epiphysitis may be present (although asymptomatic) in the dorsal spine. (4) The Mantoux test is often negative. (5) The sedimentation rate, differential count, and chest X-ray are normal.

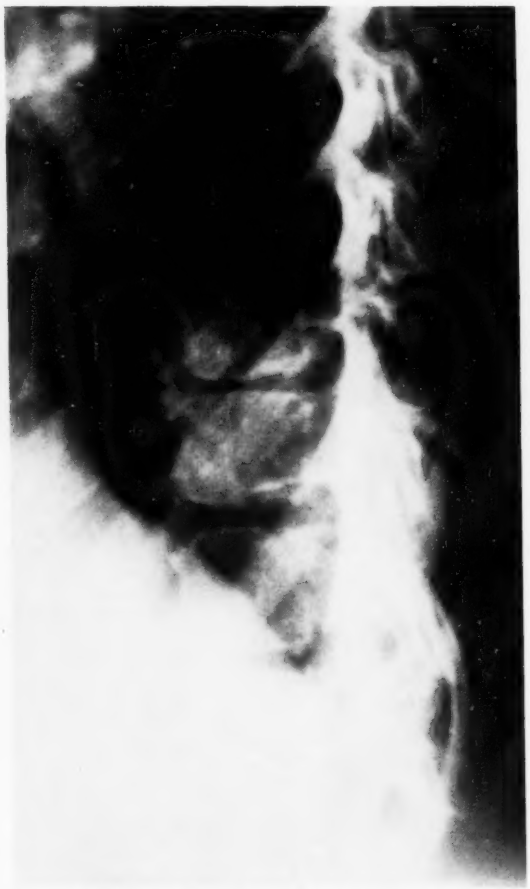

Fig. 3: Case 1 — April 23, 1956.

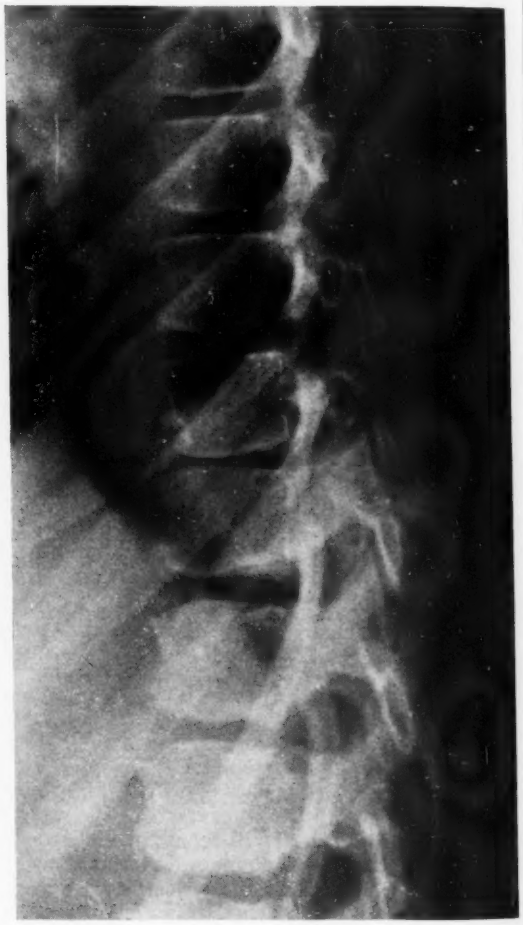

Fig. 4: Case 1 — August 28, 1956.

As shown by serial X-ray studies of these seven patients, the disease is self-limiting. The vertebral defects regenerated slowly. Complete healing took place in twelve to

sixteen months. Three patients were allowed weight bearing and normal activity. In the other four instances, bed-rest was necessary, for two to six months, with or without a plaster shell.

Case Presentations

Case 1

L.C., a girl, ten years of age, injured her spine on January 15, 1956. This injury was incurred during gymnastics at school. The patient was somersaulting over a wooden horse, and landed flat on her back. She immediately experienced pain "in the small of the back." The pain disappeared within forty eight hours, but recurred sporadically during the ensuing three months. On March 31, 1956 she consulted her physician. On physical examination the only positive finding was pain at the L.1-L.2 level on forward bending. There was no tenderness at any level. No X-rays were taken but the patient was advised to avoid strenuous exercise.

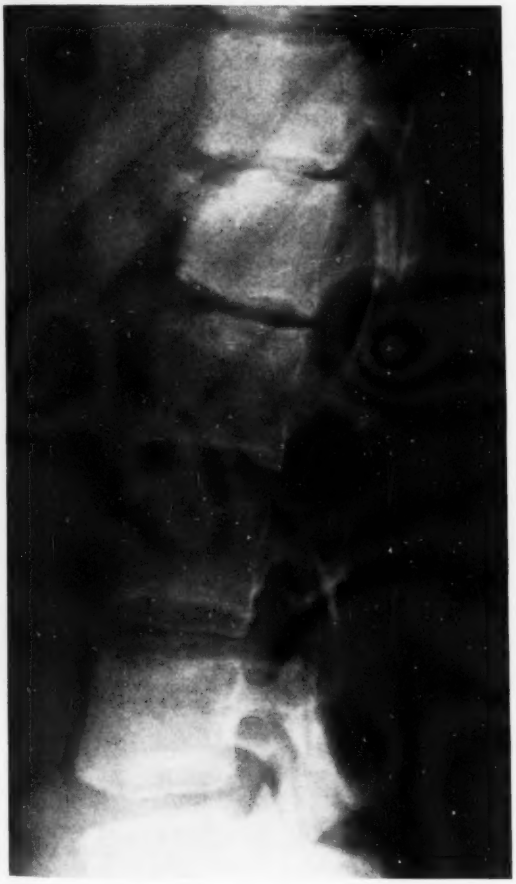

Fig. 5: Case 2 — March 3, 1955.

Pain, intermittent in nature, continued to be a feature during the next three weeks, and on April 23, 1956 the patient was sent for X-ray examination. The films showed disc space narrowing at D.10-D.11. There was no other abnormal finding in the dorsal or lumbar spine. No fracture was seen and there was no paravertebral abscess (Fig. 3).

The X-ray findings were considered to represent a traumatic disc lesion, possibly with rupture of the nucleus pulposus. The patient's Patch test was negative. Re-examination by X-ray in two or three months time was advised.

Within ten days after the taking of these films the patient's pain disappeared and has not returned. She was re-X-rayed on August 28, 1956. These films showed the D.10-D.11 disc space narrowing to be unchanged. A smooth "punched out" defect was seen at the antero-inferior margin of the body of D.10. This was typical of localized osteochondritis (Fig. 4).

The only treatment in this case was limiting the child's activities so that all strenuous exercise and sports were prohibited. She feels perfectly well and is symptomless. X-rays will be taken every three months until the healing process in the body of D.10 is complete. When last heard from in June 1957 the patient was symptom-free. She was unrestricted in her activities except for avoidence of strenuous physical training at school.

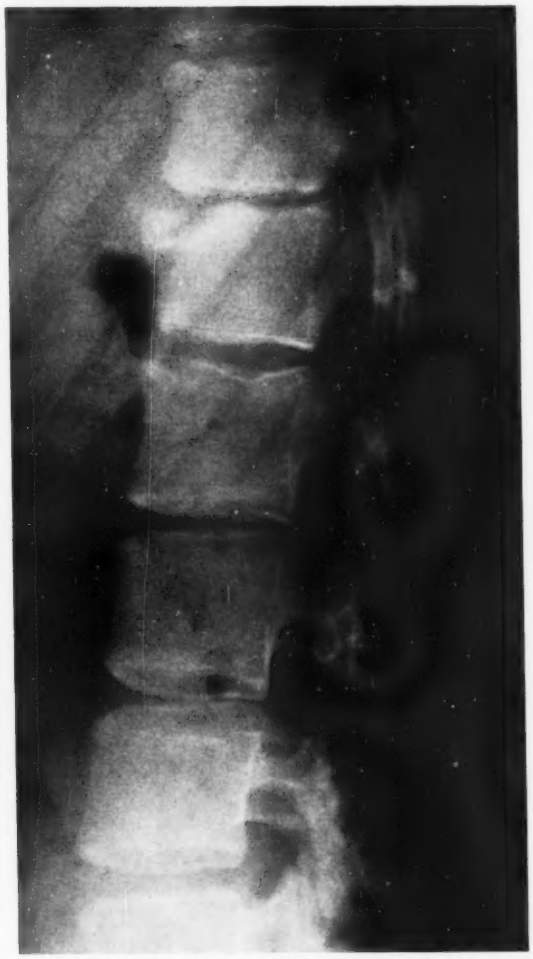

Fig. 6: Case 2 — December 13, 1955.

Case 2

L.S., a fourteen year old girl, began to complain of pain in the upper lumbar region three days after strenuous participation in Highland games. The pain was aggravated by bending and walking, and relieved by lying down. She was first examined clinically on March 1, 1955. Physical examination was negative, except for the fact that the lumbar lordotic curve was less marked than normal.

X-rays taken at the Montreal Children's Hospital on March 3, 1955 showed a typical lesion of localized osteochondritis at the antero-superior margin of the body of L.1. There was slight associated narrowing of disc spacing at D.12-L.1 (Fig. 5).

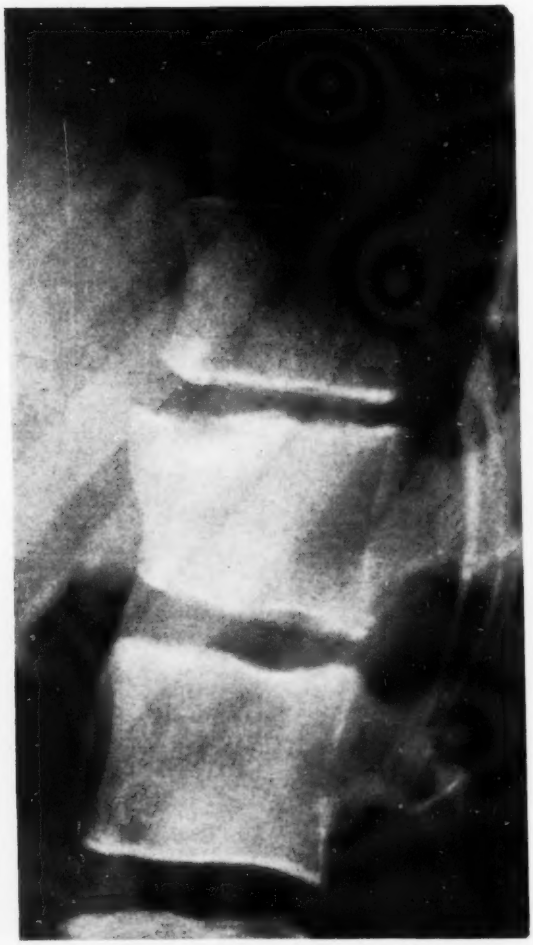

Fig. 7: Case 2 — June 20, 1956.

The only treatment was restriction of activities. The patient's pain gradually disappeared, and she was free of pain within one year. X-rays taken on Dec. 13, 1955 and June 20, 1956 show the lesion healing with slight residual deformity (Figs. 6 & 7). The disc spacing at D.12-L.1 remained slightly narrowed throughout this series of X-rays.

A progress report on this child, received on September 4, 1957, stated that she was carrying on all normal activities, including athletics at summer camp, without recurrence of her back pain.

Case 3

S.W., a sixteen year old female, presented herself for examination because the school nurse had noticed that her posture was poor. There were no symptoms. The patient had never had back pain. Physical examination was negative except for a moderate dorsal kyphosis.

X-rays taken on September 26, 1956 showed a minimal dorsal scoliosis convex to the left. They also showed typical Scheuermann's Disease in the lower dorsal area. This was associated with dorsal kyphosis. A localized lesion of osteochondritis was seen at the antero-inferior corner of the body of L.2. This appeared to be in the healed stage. The body of L.2 also showed a Schmorl's node superiorly (Fig. 8).

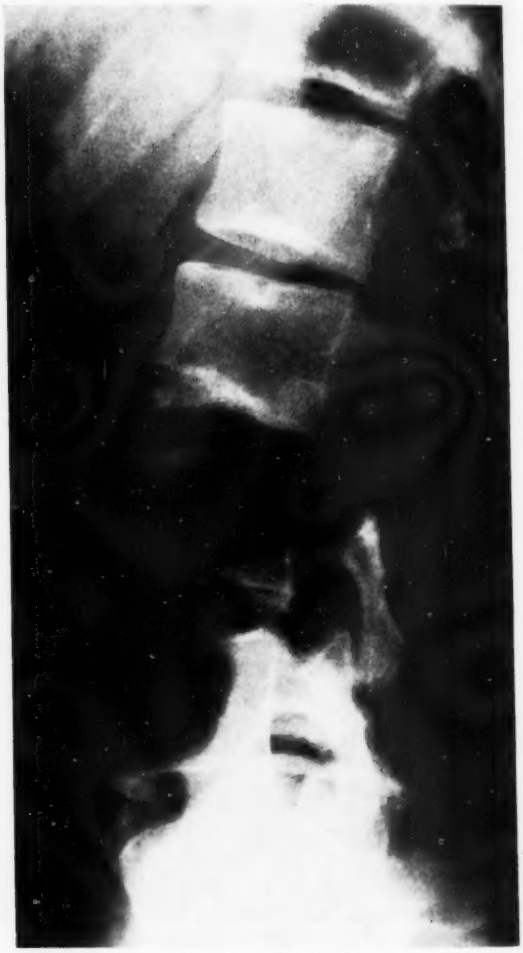

Fig. 8: Case 3 — September 26, 1956.

This patient comes in the category of a lumbar body involved by osteochondritis associated with lower dorsal lesions. The rather striking lesion of the body of L.2 is presumably an accidental finding. No history of pain can be elicited. Postural exercises are the only treatment indicated.

The latest information available on this patient came in July 1957. It indicated that she has been taking physiotherapy treatments. Her parents feel that her posture has improved slightly.

Summary

1. Osteochondritis of the dorsal spine (Scheuermann's Disease) has been reviewed in its clinical, radiological and etiological aspects.

2. Attention has been drawn to a localized form of this disease. The literature regarding this form has been briefly reviewed and theories of etiology discussed.

3. The importance of differentiating localized osteochondritis from tuberculosis of the spine has been stressed.

4. Two cases of localized lesions have been presented, together with a third case showing a lumbar body lesion associated with lower dorsal lesions.

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ROENTGENOLOGICAL EVIDENCE OF INJURY TO THE SOFT TISSUES OF THE CERVICAL SPINE

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Injuries to the supporting structures of the cervical vertebrae (ligaments and intervertebral discs) are common. These may best be discussed according to the type of deformity which is seen in post-traumatic films of the cervical spine. Four types of deformity may be descrbied. These are:

(a) Bilateral forward dislocation of the atlas;

(b) Unilateral forward dislocation of the atlas;

(c) Flexion deformity;

(d) Extension deformity.

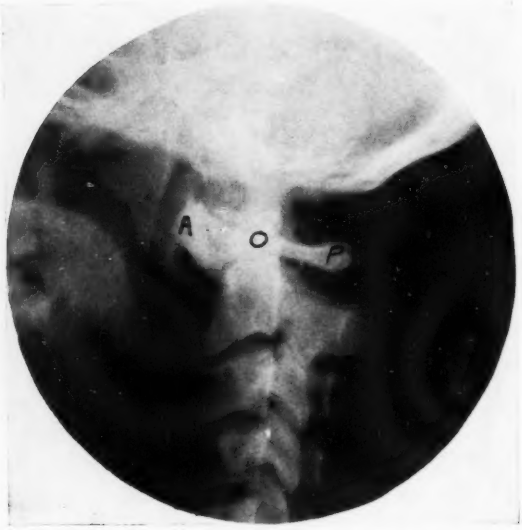

Fig. 1 — Case 1. Bilateral Dislocation of the Atlas. A— Anterior arch of atlas O— Odontoid process P— Posterior arch of atlas
M.K., male, age 20. Rugby player. Neck forcibly flexed during play, atlas forward on axis, loss of available space for cervical cord at the level of atlas 48%. No neurological signs but momentary subjective quadriplegia. Operation: Bifid arch of atlas, absence of posterior atlanto-axial membrane, underdevelopment of left half of posterior arch of atlas.

Bilateral forward dislocation of the atlas is more commonly post-infectious than traumatic in origin. The author has collected eleven cases which have been associated with injury, usually of a minor degree. (Fig. 1.) Six of these eleven cases had associated bony abnormalities in the foramen magnum or cervical spine. This, together with the relatively minor injury suffered by these patients, suggested that the transverse ligament of the atlas was underdeveloped or attenuated. The danger to the patient in this type of dislocation is the reduction in available space for the spinal cord between the odontoid process of the axis and the posterior arch of the atlas which is displaced forward. In the collected series[7] there was no correlation between the degree of displacement and the presence of cord compression.

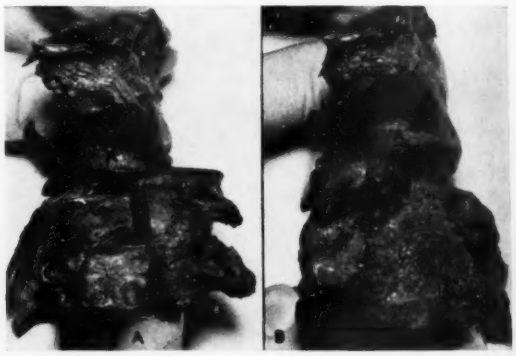

Fig. 2 — Atlanto-axial Articulation (Anatomical Specimen)
A— Atlas rotated posteriorly B— Atlas rotated anteriorly
Note biconvex nature of these surfaces.
A— Atlas E— Axis

Unilateral forward dislocation of the atlas, described in detail by Corner[2], theorizes the forward rotation of the atlas on the axis to an abnormal degree with subsequent locking of the articular facets. Close examination of these facets in anatomical specimens, however, leads one to doubt whether they can be locked. The opposing surfaces of the atlanto-axial articulation are biconvex and the margins of the inferior articulating surfaces of each lateral mass of the atlas are well rounded (Fig. 2). If one rotates the atlas on the axis until the opposing facets are completely dislocated, the rotation amounts to 60° or slightly more. At this point the cervical spinal canal is narrowed to less than one third its normal size. This would place the cervical cord in serious jeopardy. Coutts[3] states that the facets are locked just beyond the convex

ridges which traverse each articulating surface. These, however, are gradual slopes and unless there is some other factor, such as muscle spasm or the weight of the head pressing the atlas down on the axis, it is difficult to see how a dislocation could be maintained by an overlapping of these slight elevations.

The degree of dislocation encountered is one in which the normal range of movement is not exceeded; i.e., the resultant deformity as seen in the roentgenograms indicates 15° to 25° rotation which is within the normal range of movement for these joints. In a fixed anatomical specimen one can rotate the atlas on the axis 25° to 30° before one alar ligament becomes tight. Rotation beyond this point would either rupture the alar ligament or, perhaps, break the odontoid process. Coutts states 40° rotation occurs at the atlanto-axial articulation but does not specify whether he used a fixed or a fresh anatomical speciment.

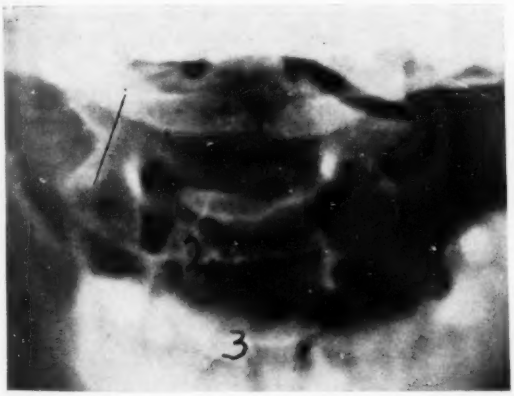

Fig. 3 — Case 2. Unilateral Dislocation of Atlas (Rotation Deformity)
Magnified view of atlanto-axial displacement.
R.M., male, age 34. Half ton of rocks fell on back (glancing blow). Sustained rotation C1 on C2 and slight forward displacement of C6 on C7. Immediate pain in nuchal area. No neurological findings. Chin rotated to left, limitation of rotation of chin to right. Traction by means of head tongs and 15 lb. weight with head extended over short mattress failed to reduce deformity.

The roentgenographic findings include an abrupt displacement of the spine of C2 to one side in relation to its normal alignment with the posterior arch of the atlas and the base of the skull (Fig. 3). The other cervical spinous processes gradually return to the midline from C2 to C7. The atlanto-axial articulation is narrowed on the side on which the atlas is in the posterior position.

The degree of disability varies. Some patients complain of limitation of rotation of the head and severe pain in the nuchal area. Others show no demonstrable limitation of rotation of the head but complain of pain on quick movement or prolonged muscular tension, i.e., driving a car. This deformity is very difficult to correct by conservative methods. Traction on the extended head by means of ice tongs and a 15 lb. weight for several days failed to reduce the dislocation in the case seen in Fig. 3. This further adds to the mystery of the anatomical lesion in this rotation deformity.

The most frequently encountered injuries result in flexion deformities of the cervical spine. Normal lateral bending films of the cervical spine demonstrate equal movement between the segments from C1 to C7 inclusive. Cramer and McGowan[4] have shown that, normally, there is minimal horizontal movement of cervical vertebrae in flexion or extension. These authors state that there is no true compression of the intervertebral discs but merely a realignment of their constituent fibres. The nucleus pulposus is the fulcrum on which the bending motion occurs. It allows the difference in height of the intervertebral discs in flexion and extension. Any arrest in this movement between flexion and extension points to a ligamentous injury with or without accompanying disc involvement.

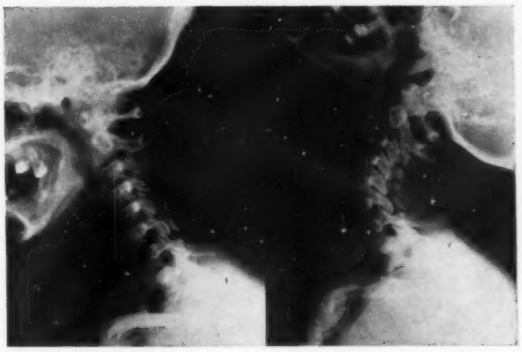

Fig. 4 — Case 4. Flexion Deformity
Tear interspinous ligament with probable impaction of intervertebral disc between C7 and D1 segments. No movement between spines of C7 and D1.
H.N., Female, age 42. Automobile accident, hit broadside. Pain in neck and down both arms in distribution of C8 nerve. Wasting of small muscles of left hand.

There appear to be two types of flexion deformity. In the first type the body of one vertebra is tilted on that of the vertebra below, producing a localized kyphosis. The spines of the vertebrae at the kyphosis are separated and films in flexion and extension

show no evidence of movement in these two segments. (Fig. 4). Ellis[5] considers that this deformity is due to rupture of the interspinous ligaments between these two vertebrae with or without impaction of the intervertebral disc. The intervertebral disc may be prolapsed in which case the spinal cord will be compressed or the patient will complain of radicular pain due to pressure on the nerve roots.

The second type of flexion deformity is one in which horizontal movement of a vertebral body on the one below is demonstrated in the bending films. In these cases, in addition to rupture of the interspinous ligament one must postulate a fracture through the intervertebral disc. Most cases are not rendered quadriplegic in spite of considerable movement at the site of injury. There is, however, no correlation between the amount of cord compression and the degree of movement of the vertebra, as demonstrated in the roentgenograms (Fig. 5).

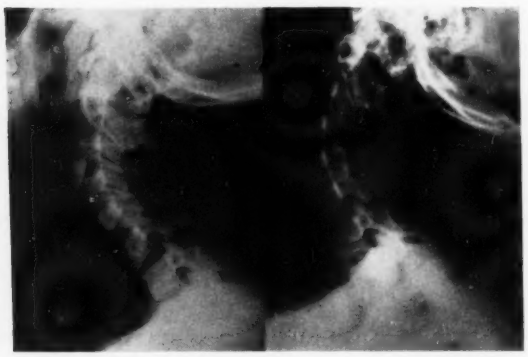

Fig. 5 — Case 6. Flexion Deformity with Marked Instability C6 C7
Injured by forced flexion of neck on mat. Immediate pain in neck. No physical finding. Four months later, deformity and instability present but no symptoms.
F.G., male, age 20. Wrestler.

The extension deformity signifies extremely serious injury. Dealing with hyperextension injuries of the cervical spine, Barnes[1] and Taylor and Blackwood[6] are of the opinion that a violent hyperextension of the cervical spine causes a rupture of the anterior longitudinal ligament. This ligament had, hitherto, been considered practically impregnable. In their cases there was no evidence of dislocation of the articular facets and lateral roentgenograms looked perfectly normal until slight extension was placed on the cervical spine and a second lateral view taken. A very small fragment of bone might be avulsed from the margin of one of the vertebrae involved. The intervertebral disc was fractured in each instance and severe damage was done to the cervical cord at the level of the fracture or, more often, cephalad to the level of the

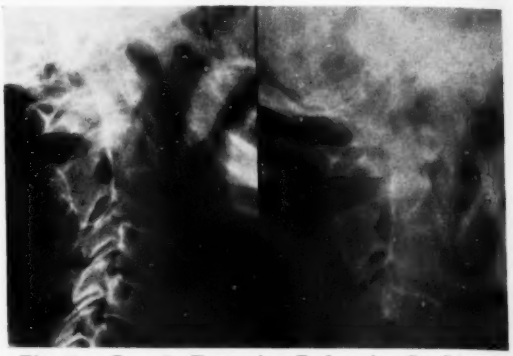

Fig. 6 — Case 7. Extension Deformity C3/C4
Retrodisplacement of C3. A— Extension B— reduced in flexion.
C2 C3 fused. Note absence of swelling of prevertebral soft tissues.
S.C., male, age 56. Fell downstairs. Immediate quadriplegia. Death in 48 hours.

Fig. 7 — Case 7. Sagittal Section Cervical Spine and Both Halves of Spinal Cord Lines point to haemorrhage into spinal cord. Note fracture inter-vertebral disc between C3/C4.

fracture. Barnes pointed out the frequency of this injury in arthritic spines. It seems to occur at a point of junction between a relatively fixed region of the spine with a mobile one.

Fig. 8 — Case 7. Close up of C3 and C4 Vertebrae. Lines indicate position of fracture through intervertebral disc.
A— Anterior longitudinal ligament ruptured P— Posterior longitudinal ligament intact. No evidence of disc protrusion.

Two cases of extension deformity of the cervical spine have been seen. Both followed a similar clinical course: immediate quadriplegia and death within 48 hours from respiratory paralysis. Post mortem in one case showed the classical findings, i.e., rupture of the anterior longitudinal ligament, fracture of the disc, haemorrhage in the spinal cord at the level of the fractured disc. There was no evidence of dislocation, nor was any bony fracture seen at post mortem examination. The disc had not prolapsed posteriorly into the spinal canal (Figs. 6, 7, 8).

Summary

1. Four types of soft tissue injury of the cervical spine have been described, based on the resultant deformity seen in the roentgenograms.

2. Unilateral dislocation of the atlas on the axis (rotation deformity) presents the greatest difficulty in diagnosis. Further enquiry should be made as to why this dislocation is maintained within the normal range of movement of these joints.

3. The degree of displacement which is seen in the roentgenogram, following flexion or extension injuries, bears no relationship to the amount of spinal cord damage which is present.

4. Hyperextension injury resulting in the extension deformity has proven lethal in a very high percentage of cases.

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THE ROENTGEN AND THE RAD AND THEIR USE IN RADIOLOGY

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Introduction

During the last 10 years there has been a great increase in the number of radiologists using both isotopes and high energy X-ray machines. With the use of these have come problems of dosimetry which have been the concern of a number of individuals as well as the International Commission on Radiological Units. We have attended meetings of this organization at Copenhagen in 1953 and Geneva in 1956 and know some of the problems faced by this organization in arriving at a system of units satisfactory to radiologists, radiation physicists and the different national groups represented on the I.C.R.U. In Copenhagen a unit of absorbed dose, the "rad", was introduced and in the years 1953-6 a number of the larger radiological centres in both England and the United States have started expressing doses in rads. In Geneva in 1956 the Commission recommended that the rad be used in radiological practice and to this end has prepared detailed discussions on the philosophy of the differences between roentgens and rads and tables to enable the radiologist to express doses in rads.[1] We shall discuss some of the reasons for the introduction of the rad and how it may be used.

One of the main difficulties in the use of the roentgen has arisen because of the dual interpretation which may be placed on its meaning. In 1937, the roentgen was defined as *the quantity of X or gamma radiation such that the associated corpuscular emission per 0.001293 grams of air, produces, in air, ions carrying 1 e.s.u. of quantity of electricity of either sign.* This has been interpreted in two different ways.

(a) Most radiation physicists and a number of radiologists have considered that the roentgen was defined as the unit which characterizes the output of an X-ray generator or characterizes the radiation coming from a radioactive source.

(b) Many radiologists and some radiation physicists have considered that the real use of the roentgen arises from the fact that it can be interpreted as a unit of energy absorbed. One roentgen given to one gram of air corresponds to an energy absorption of 87.7 ergs.* This conversion factor is constant for all energies of the radiation. One roentgen given to one gram of water corresponds to an energy absorption of about 97.7 ergs but this conversion factor varies as the energy of the radiation is changed. The conversion factor to obtain absorption in a biological material such as bone or fat varies over rather wide limits as the energy is changed.

In the early days, it was considered an advantage that the roentgen could be interpreted in these two different ways but in recent years, with the advent of high energy radiation, the dual aspect has probably caused more confusion than benefit. It was for this reason that in 1950 in London, the I.C.R.U. recommended that a unit of energy absorbed be used. Following along this line of thought, the unit, the rad, was introduced in 1953 in Copenhagen. Finally, in 1956 in Geneva, the Commission decided to try and separate clearly the two concepts and has recommended that:[1]

— the *roentgen* be a unit of exposure dose

— that is, a unit characterizing what comes from the machine, and

— the *rad* be a unit of absorbed dose equal to 100 ergs/gm characterizing the radiation absorbed in the biological material of interest.

Professor L. H. Gray, at the meeting in Geneva, gave a very useful analogy. The roentgen can be considered as a unit which measures the words flowing from the speaker's mouth, most of which pass through from one ear to the other of the members of the audience. The relatively small amount which is absorbed in the brain of the listener can be likened to the rad.

The formal definitions of exposure dose and absorbed dose are as follows:

Exposure Dose of X- or Gamma Radiation at a certain place is that characteristic of the radiation which is measured by its ability to ionize air. The unit of exposure dose of X- or gamma radiation is the *roentgen.* 1 roentgen is an exposure dose of X- or gamma radiation such that the associated corpuscular emission per 0.001293 gram of air produces, in air, ions carrying 1 electrostatic unit of quantity of electricity of either sign.

**This figure is somewhat different from former values which hvae been quoted because it is based on W = 34 e.v./ion pair. In the I.C.R.U. report, all calculations are based on this value for W.*

Absorbed dose of any ionizing radiation is the energy imparted to matter by ionizing particles per unit mass of irradiated material at the place of interest. The unit of absorbed dose is the *rad* — 1 rad is 100 ergs per gm.

You will note that the definition of the roentgen has not really been altered although it is now called a unit of exposure dose. The word *exposure* would have been preferred, rather than *exposure dose*, by most of the members of the Commission but the word *'dose'* was requested by those people who are primarily concerned with protection.

As I mentioned earlier, one runs into difficulty with the roentgen especially at high energies. In Figure 1 (curve A), is shown a plot representing the ionization measurements as the function of depth for a beam from a Co^{60} unit. The ionization rises from a small value at the surface to maximum value at a depth of about 4-6 millimeters. The absorbed

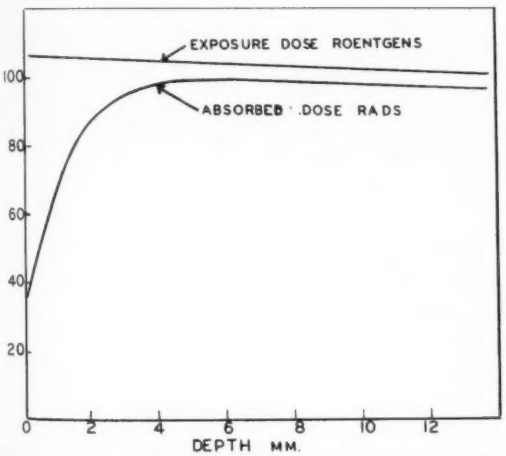

Figure 1. Graph showing the difference between "Absorbed Dose" and "Exposure Dose" in the build-up region of a Cobalt^{60} beam.

dose in rads will be proportional to this ionization curve. The scale has arbitrarily been set to read frm 0 to 100. The exposure dose, that is the quantity which characterizes the radiation coming from the unit, must have its maximum value on the surface and fall approximately exponentially as one proceeds below the surface. Thus, the distribution of exposure dose is shown by the upper curve. It has then been quite incorrect for the radiation physicist or radiotherapist to refer to the absorbed dose on the surface as so many roentgens. For 25 Mev betatron radiation, the displacement of the maximum is much deeper (about 5 cm) and the differences between these two curves are very great in the build-up region. It so happens that it is easier to measure absorbed dose directly than it is to measure exposure dose for these high energy radiations. The Commission does not feel it has enough information at the moment to recommend exactly how exposure dose should be measured for energies greater than 3 Mev. There is no abrupt change in the problem at 3 Mev since it becomes increasingly difficult to measure the exposure dose in roentgens as the quantum energy approaches very high values. The I.C.R.U. has arbitrarily regarded 3 Mev as the useful upper limit of the energy range over which the roentgen should be used. At high energies difficulties arise because of the increasing range of the electrons and decreasing range of the photons so that equilibrium is not achieved between the photon beam and the electrons.

When it comes to expressing absorbed dose from a beta emitting radio-active material, in terms of the concentration in millicuries/gm present at the point of interest, one merely multiplies the number of disintegrations per second by the mean energy of the disintegration to obtain the energy absorbed in ergs/gm. If this is divided by 100, we obtain the dose in rads. For a number of years, the unit, the rep, was used for this type of dose estimation. The definition of this unit was never accepted widely and as a result the unit meant a different amount of absorbed energy for different people. Its use is no longer recommended.

Finally, the rad should be of value in estimating absorbed doses in bone or fat. Before the introduction of the rad, the radiologists had no unit in which they could express the absorbed dose in bone or fat. He was always aware of the fact that the absorbed energy in the bone might be several times that estimated from standard depth dose tables but he did not have a unit in which he could express this dose. It is hoped, with the introduction of the rad, that it will focus the radiotherapist's attention on this problem.

In the following table, some conversion factors from roentgens to rads, taken from the I.C.R.U. report, are given for a number of types of radiation. The first column gives the tube potential, the second the filter, the third the half value layer and the fourth the spectrum. The spectra of the radiations for some of the cases were measured, for others have been calculated using Kramer's formula.[2] The last three columns give the conversion factors for water, muscle and bone. For water and muscle, the conversion factors do not vary much from the average value of about 0.96.

Table I

$\overline{f}$, (rads per roentgen) for Water, Muscle and Bone, for Various Primary X-ray Spectra (I.C.R.V.)

Tube potential (kv)	Filter	(HVL) (mm)	Spectrum	$\overline{f}$ — ($\overline{rad/r}$)		
				H_2O	Muscle	Bone
100	0.18 Cu	0.25 Cu	measured	0.91	0.94	3.10
200	0.20 Cu	0.5 Cu	Kramer's	0.94	0.95	2.05
250	0.17 Cu + 3.0 Al	1.0 Cu	Kramer's	0.95	0.95	1.76
250	0.9 Cu + 3.0 Al	2.0 Cu	Kramer's	0.96	0.96	1.42
280	—	2.5 Cu	measured	0.97	0.96	1.22
400	—	4.16 Cu	measured	0.97	0.97	1.11

The data of Table I is put in more useful form in Table II where the conversion ratios are given for a number of different half value layers.

Table II

Conversion Ratios, rads roentgen, for the primary X-ray spectrum with half value layers shown in the first column. Data taken from the report of the I.C.R.U.

Half value layer mm Cu.	$\overline{f}$ rads per roentgen		
	Muscle	Water	Bone
0.5	0.950	0.942	2.05
1.0	0.955	0.952	1.74
2.0	0.961	0.965	1.35
3.0	0.964	0.972	1.14
4.0	0.966	0.975	1.10

The values of f given in Tables I and II are based on the primary spectrum only. In actual fact, there is always present the contribution of the softer scattered radiation, the proportion of which changes with depth and area of field. When further data on the scattered spectra is available, it should be taken into account. However, it is evident from the rather slow variation of f, with half value layer shown in Table II, that inclusion of the scattered component will not alter the results very much.

The way these tables can be used in practice will now be illustrated by a simple example. Suppose the patient is to be treated with 280 KV, F.S.D. 50 cm, H.V.L. 2.0 mm Cu with a field size of 100 cm^2 and that the tumor is 6 cm below the skin surface. For this radiation standard depth dose tables give a back-scatter factor of 1.31 and a depth dose of 61.0%. For this radiation f muscle = 0.961, f bone = 1.35. Suppose the exposure dose at the skin position without backscatter is 1000 roentgens (this corresponds to the "dose in air") then we calculate the following:

Absorbed dose in soft tissue at the skin surface =

$1000 \times 0.961 \times 1.31 = 1260$ rads

Absorbed dose at 6 cm depth in soft tissue =

1260×0.61 rads $= 768$ rads

Absorbed dose at 6 cm depth in bone =

$1000 \times 1.31 \times 1.35 \times 0.61 = 1080$ rads

Due to the finite range of the electrons in material the absorbed dose will not suddenly alter from 768 rads to 1080 rads at a soft tissue bone interface at the 6 cm depth. A 250 Kev electron has a range of about 0.5 mm in tissue so that the dose will change from one value to the other over a distance of approximately 0.5 mm.

It is likely that for each treatment field of the X-ray machine the radiation physicist will record the absorbed dose rate in soft tissue at the skin surface in rads/min. The isodose curves may then be used directly to obtain the absorbed dose rate at any other point in soft tissue. A conversion factor is then applied, if the point of interest is in bone or fat. The changing over of a department from measuring in roentgens to rads requires some reorientation in thinking on the part of the radio-therapist. In soft tissue the former dose of 5000 roentgens would now be prescribed as a dose of about 4800 rads in soft tissue.

There is still some doubt as to the actual conversion factors because of uncertainties in W (the energy required to produce an ion pair) and S (the stopping power). However, the Commission, using the best available values, has published a detailed report on the conversion factors from roentgens to rads.[1] It is hoped that all radiotherapy departments using the rad will use the recommended conversion factors so that no ambiguity will result from the use of different factors. These conversion factors will be continually held in review by the Commission in the light of the best information which is available. The Commission feels that conversion from roentgens to rads can be carried out without too much difficulty and that the conversion will enable radiotherapists and physicits to think more logically concerning radiation dosimetry. They hope that the system will be widely used in practice.

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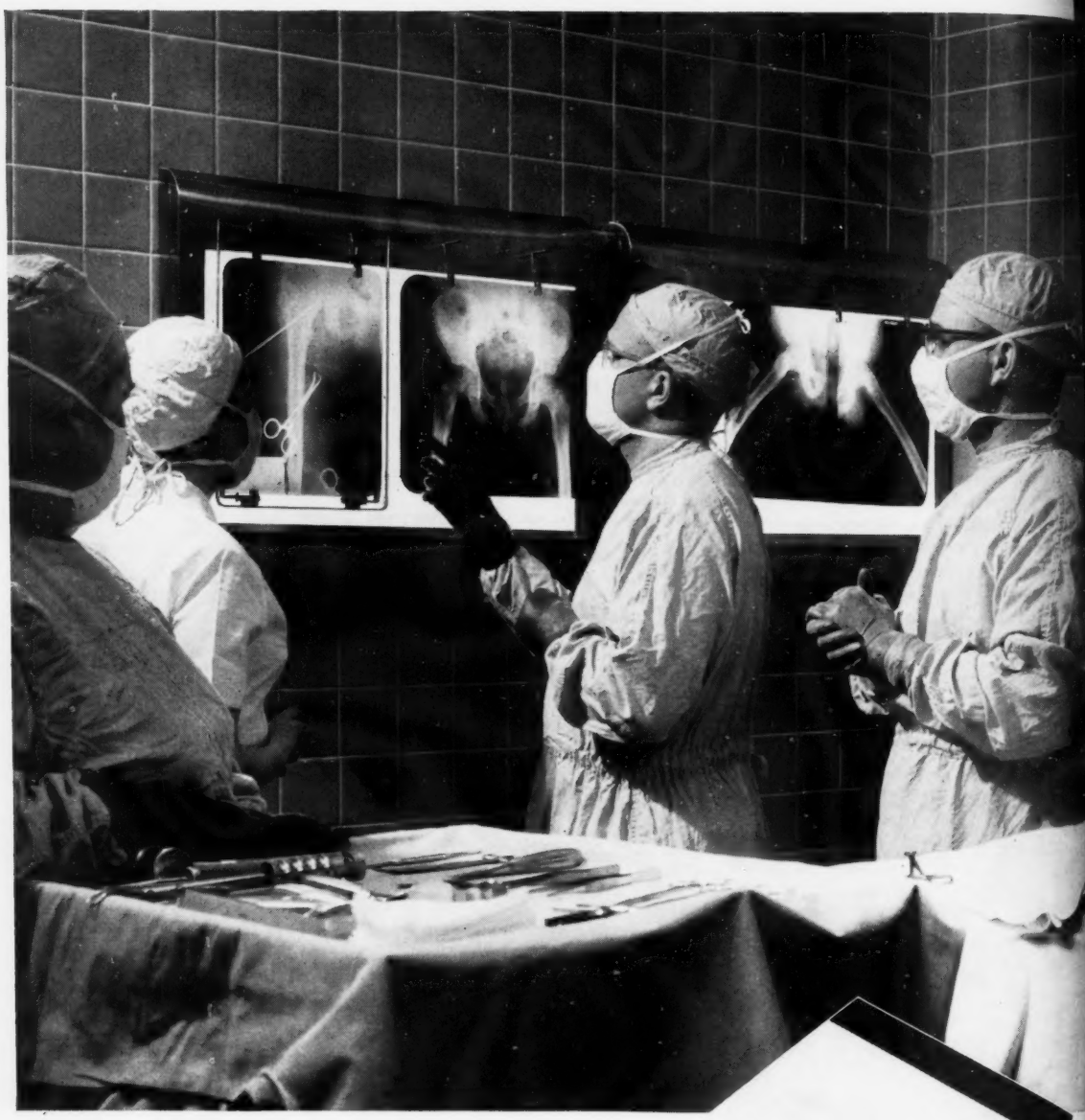

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